

Does ethanol decrease the GCS in TBI patients?

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Forord

Min prosjektoppgave er skrevet som en artikkel med håp om senere publisering i et vitenskapelig tidsskrift. Følgelig er teksten skrevet på engelsk, og formatet er lagt opp deretter. Endelig er dette mitt utkast, hvilket sikkert vil bli modifisert før eventuell innsending.

Jeg vil takke veileder professor i nevrokirurgi Eirik Helseth for kyndig veiledning og det kliniske sommerstipendiat jeg ble gitt ved nevrokirurgisk avdeling UUS sommeren 2009 til arbeid med oppgaven. Jeg vil også takke overlege nevrokirurgi Pål Rønning som har hjulpet meg gjennom hele prosessen - fra konkretisering av problemstilling til veiledning med alt fra engelsk språkføring, faktiske tekstbidrag, oppbygning av artikler til faglig diskusjon og løsning av statistiske problemstillinger.

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Does ethanol decrease the GCS score in TBI patients?

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Key words: Traumatic brain injury, Glasgow Coma Scale, Ethanol, Alcohol

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Abstract:

Background: 20-50% of patients with traumatic brain injury (TBI) are under the influence of ethanol at hospital admission. The Glasgow Coma Scale (GCS) is used in the initial assessment of these patients, directing further diagnostic evaluation and therapy. Therefore, we investigated whether ethanol has an impact on the initial GCS assessment in TBI patients.

Methods: From the trauma registry at Oslo University Hospital Ullevål (OUHU), we included patients with blunt head injury between 14 and 80 years old, with known head Abbreviated Injury Score (AIS), blood ethanol concentration (BAC)- and GCS score at admission. 841 patients were included and categorized by BAC into an ethanol influenced- (n=474) and a non-influenced group (n=367). The two groups were compared by several admission and outcome variables.

Results: Mean BAC in the two groups were 0 ‰ and $2,1 \text{ ‰} \pm 0,1 \text{ ‰}$. The groups were equal with regards to mean GCS ($11,7 \pm 0,41$ vs. $11,3 \pm 0,38$, $p=0,151$) and within all AIS categories except for AIS=2. Uni- and multivariate regression analyses together with matched bootstrapped- and robust linear regression were unable to show a significant impact of ethanol on GCS. The groups were similar regarding gender, age, length of stay, ventilator requirements and presence of hypotension at admission. However, ethanol influenced patients sustained less severe injuries, had better survival, fewer days in the ICU and lesser need of intubation.

Conclusion: Ethanol intoxication does not reduce the GCS score to a clinically significant degree and reduced GCS score should not be attributed to ethanol intoxication.

Key words: Traumatic brain injury, Glasgow Coma Scale, Ethanol, Alcohol

Introduction:

The extent to which the ethanol intoxication influences the Glasgow Coma Scale (GCS) score is debated, and studies have shown that as many as 20-50% of patients entering the emergency department with traumatic brain injury (TBI) are under the influence of ethanol (1). At Oslo University Hospital Ullevål (OUHU), the annual incidence of hospital-treated TBIs is 83.3/100.000 (2). The GCS is an important parameter in clinical practice (3). The initial GCS score of TBI patients is used in algorithms directing further management (4;5). The GCS is also used as a prognostic marker, either alone or in combination with for example the head Abbreviated Injury Scale (AIS) and Injury Severity Score (ISS) (6).

Given that ethanol decreases GCS, guidelines and algorithms based on this score could cause the TBI of influenced patients to be treated as more severe than it would actually merit. For the patient, the consequence could be increased risk of acquiring iatrogenic injuries because of unnecessary interventions and hospitalization. For the hospital, economical and administrative implications could be the consequence of resulting superfluous diagnostics and treatment. On the contrary, if diagnostics and treatment are delayed because the decreased GCS score is attributed to the effect of ethanol, it might have consequences for prognosis, possibilities for treatment and might cause severe permanent injury or even death (7).

Sperry et al. investigated in 2006 if ethanol decreases GCS in 1,075 blunt TBI patients admitted to a level-1 trauma center (8). In 2007, Stuke et al. published a paper on the same topic on 108,929 TBI patients admitted to trauma centers in USA (9). Both studies concluded that ethanol does not decrease GCS.

Although Stuke and Sperry have investigated the influence of ethanol on GCS assessments, this topic remains of great clinical importance, and evidence from other institutions and nations is relevant in order to confirm these findings and contribute to a greater evidence base. We therefore wanted to investigate the effect of ethanol on GCS in a hospitalized TBI population.

Material and methods:

Oslo University Hospital Ullevål (OUHU) serves as the regional trauma care facility for approximately 2.5 million people. Geographically the catchment area for the trauma facility is 110,000 km². Patients with severe trauma are most often transported directly to OUHU, while seemingly less severely injured patients are treated at other hospitals in the region and transported to OUHU if needed after consultation. Patients with head injury suspected to be in need of urgent neurosurgical care, are transported directly to OUHU. The trauma registry prospectively includes all patients with Injury Severity Score (ISS) ≥ 10 whether they are admitted to OUHU directly or via a local hospital within 24 h after injury. Moreover, the registry also includes all patients admitted under the auspices of the trauma team, and/or penetrating injuries towards the torso, and/or proximal to elbow or knee, irrespective of ISS. The trauma team is alarmed upon admission of patients obviously severely injured, if unstable (circulatory/respiratory instability or reduced level of consciousness), victims of high-energy trauma, and in other situations with a high index of concern.

The trauma registry contained 4,038 patients admitted with TBI in the period of 2002-2007 to Oslo University Hospital Ullevål (OUHU). In the registry, TBI was defined as a head trauma with a GCS < 15 and/or a head AIS > 1 , without asphyxia from drowning or an additional

medical condition. The values for all variables were prospectively entered into the database by two experienced registrars with trauma team experience who are formally educated in AIS coding (AIS '98).

Figure 1 shows the inclusion criteria and the inclusion process, which resulted in 841 patients fulfilling the inclusion criteria. These patients were divided into two groups based on the presence of ethanol in blood sample drawn at admission. The non-influenced patient (NIP) group with blood alcohol concentration (BAC) = 0‰ consisted of 367 patients, while 474 patients with BAC > 0‰ comprised the influenced patients (IP) group.

The head abbreviated injury scale (AIS) was used as a marker for anatomical injury in the two groups. It is an anatomical marker based on either neuroradiological- or peroperative findings. AIS is composed of six categories where category 1 reflects minor injuries and category 6 represents maximal and usually lethal injuries (10). We used the injury severity scale (ISS) as a parameter for total injury severity in the patient. ISS is calculated by adding the square of the three highest organ AIS scores. It ranges from 0 – 75 where 75 represents maximum injury. An AIS = 6 in any organ system is automatically assigned a score of 75 (11).

We formulated the following H_0 : $GCS_{IP} = GCS_{NIP}$. First, the groups were compared by several variables. With two independent groups, *continuous* variables were analyzed with Student's t-test when normally distributed and with Mann-Whitney test when the distribution was skew or with unequal variances. For *categorical* variables, we used Chi-square and Fischer's exact test. Ordinary least squares regression was performed but revealed heteroscedasticity. This was circumvented by utilizing both robust regression methods and bootstrapping of the coefficients. Stratified for AIS more than 10.000 random samples where

made from drawing data from the categories of 30 day survival and age, parameters where there were an significant diffence between the groups, to calculate 95 % confidence intervals for the effect of BAC on GCS.

A p-value < 0,05 was considered statistical significant, and difference in GCS ≥ 1 was considered clinically relevant. SPSS v15.0 and R v2.11 was used for the analyses.

Results:

The mean BAC in the two groups were 0,0 ‰ and 2,1 ‰ \pm 0,1. There were no significant differences in age or gender, with men dominating our study population. The groups were equal considering days admitted to hospital, days on ventilator and presence of hypotension on admission (defined as systolic blood pressure (SBP) < 90mmHg). IP had lower ISS, lower 30 days mortality rates, needed fewer days in ICU and had less need of intubation. IP had also a greater base excess deficit at admission. Regarding mechanism of injury, IP sustained fall and violence related injuries significantly more often than NIP. Motor vehicle related injuries were more prevalent in the NIP group. See table 1 for summary. There were roughly the same number of IP and NIP in the categories of AIS = 1, AIS = 2 and AIS = 5, while AIS = 3 and AIS = 4 were dominated by IP (fig. 2).

The mean GCS score at admission was not significantly different between the two groups. When comparing the means for the components of the GCS, only the eye component was statistically significantly different with IP scoring lower than NIP (2,89 \pm 0,15 vs. 3,13 \pm 0,15, p = 0,018), see table 1. When we stratified the GCS scores against the head AIS, we found that the GCS scores for IP and NIP were equal for every AIS category except in AIS =

2. In this category, IP had a statistical and clinical significant lower GCS score than NIP ($12,23 \pm 0,64$ vs. $13,61 \pm 0,47$, figure 3). In addition, in the group of AIS = 2, IP had a significantly lower score for all components (motor; $5,09 \pm 0,29$ vs. $5,64 \pm 0,21$, verbal; $3,81 \pm 0,29$ vs. $4,36 \pm 0,24$, eye; $3,13 \pm 0,22$ vs. $3,57 \pm 0,16$).

No difference was found when comparing IP with $BAC \geq 3,0\%$ with NIP, $p=0,81$. The univariate regression analysis showed no significant impact of increasing BAC on GCS. In the multivariate model with an adjusted $r^2 = 0,625$, BAC had a $\beta = -0,34$, $p<0,001$. See table 2 for all data and model summary. Generalized additive analyses showed aberrancy only within the AIS = 2 group, illustrating that a linear model is acceptable but not completely optimal (figure 3). The matched bootstrapped regression model and the robust linear regression found a statistical significant effect of BAC on GCS only within the AIS = 2 group ($[-0,9, -0,23]$ and $[-0,52, -0,09]$ respectively, see table 3).

Discussion:

Our results indicate that ethanol does not lower GCS score in TBI patients to a clinically significant extent. However, there is a difference for patients with AIS = 2. The linear regression models showed that ethanol poorly predicts the GCS; the univariate analysis was unable to show any isolated effect of BAC on GCS, however in the multivariate model, BAC as predictor had a $\beta = -0,34$ ($p< 0,001$). This illustrates that a increase in BAC-level from 0,0 ‰ to $\sim 3,0$ ‰ is needed before a change in GCS greater than 1 is obtained. In addition, neither the matched bootstrapped- nor the robust linear regression analyses revealed any obvious effect of ethanol on the GCS score.

NIP in our study group had lower survival rates, longer stays in the ICU and greater requirements for intubation. This is probably explained by NIP dominating the AIS = 5 group with the most severe injuries, the majority due to high energy motor vehicle accidents. The lower base excess in IP is most likely due to the metabolic acidosis induced by ethanol consumption (12).

We find the significant effect of ethanol on GCS in the AIS=2 group unconvincing. First, if ethanol decreases the GCS score in TBI patients, one might expect to see some differences in the other AIS categories as well. We only found a difference in the AIS = 2 group, suggesting that this might be a random finding. Secondly, one should consider that ethanol testing was done selectively on admission and that patients with unknown BAC status were excluded in this study. The possible selection bias resulting from this could cause a falsely low GCS in NIP. Cherpitel et al. found that health care professionals poorly identify patients with moderate BAC levels, but that patients with a high BAC usually are identified (13). This could explain the high BAC in IP. Finally, the difference could be an expression of inter-rater-variance and uncertainty in GCS assessment, which also could be influenced by the physicians' expectations and uncertainty during examination of IP (14). It is reasonable to assume that such an effect would be greatest for conscious though injured patients, like those in AIS=2.

The ethanol tolerance in the IP group could be higher than in the general population, hiding an actual difference in GCS score that would be discovered if we had analyzed individuals with comparable ethanol tolerance (1). However, the high mean BAC of 2,1 ‰ held together with no measurable difference in GCS comparing IP with BAC > 3,0 ‰ to NIP disavows this

effect. Ethanol concentrations at these levels would probably cause impairment even in the most tolerant individuals (1;15).

Another consideration is the lack of knowledge regarding the IP group and their pre-morbid state. This group is likely to contain more ethanol dependent patients, and common pathological changes in this group (16), for example cortical atrophy and neuropathy, could influence both the AIS score and the GCS assessment. But, with average age of 37,4 in IP and 36,1 in NIP, we consider this effect to be small.

In the above, we only considered ethanol, disregarding the presence of other intoxicants. However, one could assume this effect to be higher in the IP group, knowing that coincidental drug/narcotic and ethanol abuse is common (1).

Finally, all data represent a single institution experience that could result in demographical idiosyncrasies. However, OUHU being a regional trauma center, receiving patients from all eastern Norway, helps to minimize this effect. This also points to another limitation; our material consists of patients admitted to the trauma unit with a high index of suspicion for severe injury and might not generalize to emergency rooms where the prevalence of head-injury is less. The considerations regarding the study population affects the extent to which we can generalize our findings; it should be emphasized that they relate to a hospitalized population. Furthermore, our conclusion might be correct regarding the group as a whole, but not when applied on an individual level.

The patients were prospectively entered into the database, but we collected the lab data in retrospect, which increases the risk of not discovering confounding factors. However, we have a large sample size and relatively consistent findings.

As mentioned in the introduction, the study done by Stuke et al. in 2007 with 108,929 patients collected from trauma centers nationwide in USA failed to show reduced GCS in middle aged patients with blunt TBI under ethanol influence (9). The only exception was found in patients with a mild head injury (AIS = 0 or AIS = 2) but a high total injury burden (ISS > 25). Sperry et al. examined the same matter in 2006 with 1,075 blunt TBI patients from a level-1-trauma center (8), where AIS = 1 and AIS = 2 were merged into one group labeled “concussion”. They found lower GCS scores for IP in the AIS = 5 category for patients with ISS > 18 and for patients with SBT > 90 mmHg. Apart from this, NIP and IP had the same GCS. Both studies concluded that ethanol does not lower GCS to a clinically significant extent.

No previous studies before Stuke’ and Sperry’ had investigated the impact of ethanol on GCS directly. However, previous investigations indirectly described the relation between ethanol and GCS. Some studies found a decreasing effect of ethanol on GCS (17-19). Jagger et. al found a effect when BAC levels exceeded 2,0 ‰, and Galbraith et al. suggested that a decreased GCS in patients with BAC levels < 2,0 ‰ should not be explained by ethanol (20;21). Dunne et. al found a statistical significant, but clinical ignorable effect of ethanol on GCS (12), and finally there were studies stating that ethanol did not decrease the GCS score or consciousness (22;23).

In recent studies it has been suggested that ethanol might have a protective effect concerning mortality in patients with TBI, but the effect has not been significant and the mechanism

remains poorly understood (24;25). The increased 30 days survival among IP in our results is, as already discussed, most likely a result of fewer patients with AIS = 5. Nevertheless, more knowledge about the biochemical and physiological effects of ethanol in TBI patients would be of great interest given their tight relationship. In addition, investigations on how GCS is affected by drugs and narcotics, by different drugs and ethanol combined and in patients with comparable ethanol tolerance would be of interest in the context of emergency care and management of TBI patients.

We conclude that ethanol does not cause a clinically significant reduction in GCS score in TBI patients. Hence, diagnostic and treatment of these patients should not be delayed because of a suspicion of ethanol influence.

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Table 1: The group characteristics for various admission variables and distribution by injury mechanism for NIP and IP.

	<i>N</i>	<i>NIP</i> <i>Mean and 95% C.I.</i>	<i>IP</i> <i>Mean and 95% C.I.</i>	<i>P-value</i>	
BAC in permille	841	0,0 ± 0	2,1 ± 0,1	<0,001	†
Gender (% male)	841	81 % ± 4%	84 % ± 3 %	0,241	Δ
Age at injury (years)	841	36,1 ± 1,7	37,4 ± 1,4	0,261	†
Injury Severity Scale (ISS)	841	19,0 ± 1,5	15,5 ± 1,0	0,010	•
Survival 30 days (% yes)	841	92 % ± 3 %	95 % ± 2 %	0,035	Δ
Length of stay (days)	841	6,4 ± 0,9	5,6 ± 0,8	0,210	†
Days in ICU	769	4,7 ± 0,8	3,4 ± 0,6	<0,001	•
Days on ventilator	325	7,4 ± 1,3	5,8 ± 1,2	0,082	†
Intubated prehospital or at ED (% yes)	841	44 % ± 5%	37 % ± 4 %	0,035	Δ
Systolic BT in ED (mmHg)	809	137,6 ± 2,8	127,6 ± 2,1	<0,001	†
Hypotension (% SBP<90mmHg)	841	3 % ± 2 %	3 % ± 2 %	0,932	Δ
BE	670	-1,3 ± 0,4	-3,4 ± 0,4	<0,001	†
GCS at admission	841	11,7 ± 0,41	11,3 ± 0,38	0,151	†
GCS motor component	560	5,15 ± 0,18	4,93 ± 0,18	0,163	•
GCS verbal component	559	3,77 ± 0,19	3,51 ± 0,18	0,053	†
GCS eye component	559	3,13 ± 0,15	2,89 ± 0,15	0,018	†
By injury mechanism;					
Motor vehicle related	837	266 (73 %)	185 (39 %)	<0,001	Δ
Fall related	822	65 (18 %)	185 (40 %)	<0,001	Δ
Violence related	815	22 (6 %)	85 (19 %)	<0,001	Δ
Self mutilation	826	11 (3 %)	9 (2 %)	0,330	Δ
Work related	839	19 (5 %)	1 (0 %)	<0,001	Δ
Sport/recreational	840	8 (2 %)	5 (1 %)	0,191	Δ
Other	841	4 (1 %)	0 (0 %)	0,036	λ

• Mann-Whitney

Δ Chi-square

† Student t-test

λ Fischer's exact test

IP: Patients influenced by ethanol

NIP: Patients not influenced by ethanol

Table 2: The results from the regression analyses.

Univariate regression analysis

Predictor	Beta \pm SE	95 % C.I.	P-value
BAC (in ‰)	-0,130 \pm 0,117	[-0,36, 0,1]	0,264

Multivariate regression analysis

BAC (in ‰)	-0,343 \pm 0,072	[-0,484, -0,202]	<0,001
Age at injury	0,018 \pm 0,006	[0,007, 0,030]	0,002
Intubated prehospital or at ED (1=Yes, 2=No)	5,204 \pm 0,203	[4,805, 5,602]	<0,001
Systolic BP < 90 mmHg (1=Yes, 2=No)	1,431 \pm 0,505	[0,440, 2,422]	0,005
Head AIS	-0,915 \pm 0,078	[-1,068, -0,761]	<0,001
(Constant)	2,638 \pm 1,077	[0,524, 4,752]	0,015
Adjusted R ² = 0,625	ED: Emergency department	Dependent variable: GCS.	

Table 3: The results from the matched bootstrapped- and the robust linear regression analyses.

<i>Matched Bootstrapped</i>				<i>Robust linear regression</i>		
AIS	Intercept with C.I.	BAC with C.I.	Age with C.I.	Intercept with C.I.	BAC with C.I.	Age with C.I.
1	14.77 [13.68, 15.7]	-0.25 [-0.63, 0.03]	-0.01 [-0.04, 0.02]	15 [15, 15]	0 [0, 0]	0 [0, 0]
2	12.76 [11.62, 13.93]	-0.56 [-0.9, -0.23]	0.02 [-0.02, 0.05]	13.95 [13.28, 14.63]	-0.3 [-0.52, -0.09]	0.01 [-0.01, 0.02]
3	10.54 [8.94, 12.13]	-0.09 [-0.57, 0.42]	0.03 [-0.01, 0.07]	10.43 [8.82, 12.04]	-0.04 [-0.54, 0.45]	0.03 [-0.01, 0.06]
4	9.36 [7.67, 11.1]	-0.36 [-0.92, 0.2]	0.04 [0.01, 0.07]	9.09 [7.39, 10.79]	-0.22 [-0.76, 0.33]	0.04 [0.01, 0.08]
5	4.48 [2.79, 6.75]	0.19 [-0.52, 0.78]	0.03 [-0.02, 0.08]	4.26 [2.94, 5.58]	-0.07 [-0.67, 0.52]	0.04 [0, 0.07]

Figure 1: The inclusion process from the trauma registry of OUHU. From the 4039 patients in the registry, only patients with known scores on each criterion were enrolled.

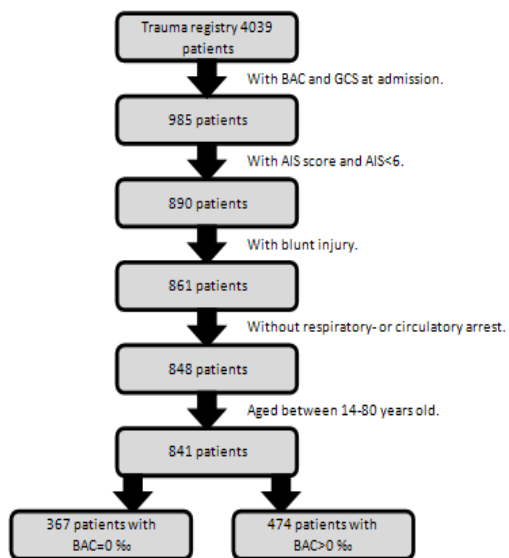


Figure 2: The number of patients for IP and NIP within each head AIS category.

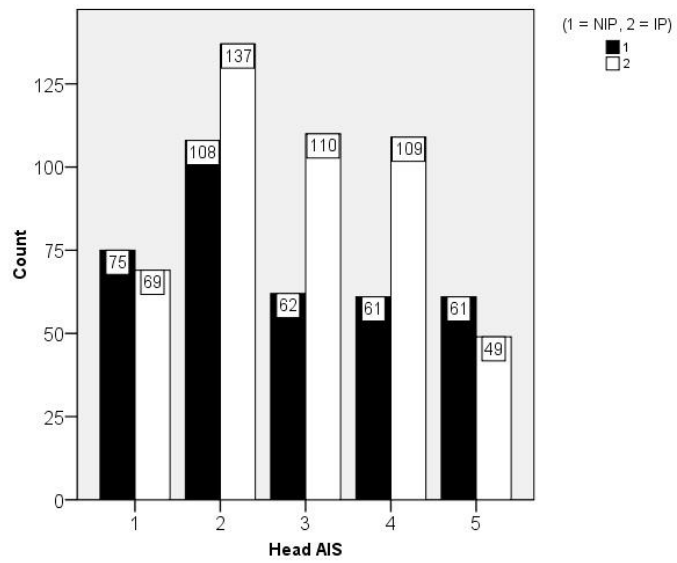
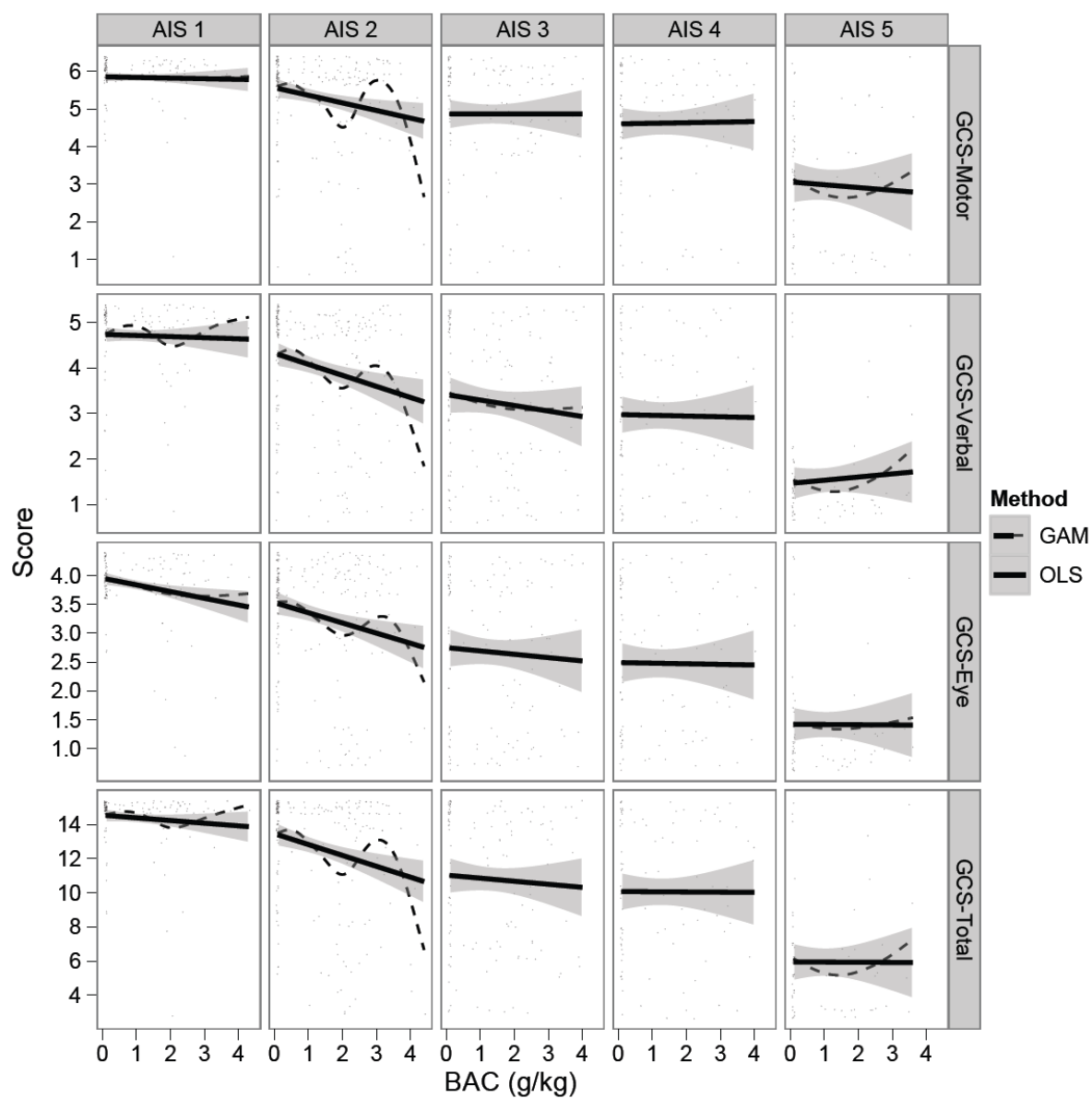


Figure 3): Mean GCS score and each of its components against head AIS for different levels of BAC.



GAM=generalized additive model, OLS=ordinary least square